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An Accidental Overspill: A Case Report of Fatal Paraquat Poisoning by Dermal Absorption

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Abstract

Introduction: Paraquat dichloride is a widely used, highly toxic herbicide which is sold as a brownish concentrated liquid of 10–30% strength. Due to easy availability and no specific antidote, numerous fatal cases of paraquat poisoning by ingestion have been reported accidently or with a suicidal intent.

Case description: A 30 year old alcoholic man who unintentionally consumed poison in an inebriated state leading to spillage over the unhealed skin lesions over neck and chest area, who presented to hospital emergency 5 days after the exposure with complaints of breathlessness and reddish skin lesions and subsequently died over the course of treatment.

Discussion: The findings of this case highlight fatal systemic effects caused by accidental spillage over dermal lesions leading to respiratory and renal failure. This signifies the magnitude of dermal absorption and the measures to avoid a minimal exposure turning into a hazardous event.

Keywords: Paraquat, pulmonary fibrosis, skin absorption, herbicide, renal failure, respiratory insufficiency.

Introduction

Plant Protection Ltd. introduced paraquat (dichloride 1, 1-dimethyl-4, 4-bipyridylium) to the market in 1958, and it is today the third most extensively used herbicide. The chemical is mostly safe when used as directed, however misuse has resulted in a considerable number of deaths.[1] The Central Insecticide Board and Registration Committee (CIBRC) in India has approved a 24 percent formulation that is labelled as highly dangerous and sold under the name Gramoxone. It is mostly consumed in four Indian states: Punjab, Goa, Maharashtra, and Kerala.[2] It's extremely dangerous to people, and it's been linked to a number of incidents of acute poisoning. Pulmonary fibrosis owing to lipid peroxidation is a primary symptom of its poisoning, and the majority of cases occur from accidental or intentional intake, resulting in death[3]. However, a few reported cases show that paraquat may be absorbed through skin lesions and this dermal route can cause systemic toxicity.[4, 5]

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We hereby present such a case brought for autopsy with an alleged history of unknown poison intake. The poison was later discovered to be paraquat after proper history taking and as per circumstantial evidence.

Case Presentation

A 29-year-old man, farmer by profession and a known alcoholic presented to the hospital causality with complain of breathlessness since four days along with multiple episodes of blood stained vomit since one day. On initial examination pulse rate was 102/min, Blood pressure 130/80 mmhg, Respiratory rate 53/min, SpO_2 on admission was 60% on room air and 85% on oxygen. Pallor and icterus were highly evident. Bilateral lungs showed crepitation. Abdomen was not distended but showed mild tenderness in epigastric region with normal bowel sounds.

Patient was wearing a foul-smelling shirt. Removal of the shirt exhibited ulcerated skin lesions over the front of neck and chest with minor lesions over the lower lip. Some areas of the body showed multiple whitish lesions with a central clearing particularly over the groin area and inner aspects of thighs. Chest X-ray showed findings compatible with ARDS and the clinical findings in hospital record was documented as acute lung injury, acute kidney injury and leucocytosis. Patient was managed in intensive care unit, he was intubated on arrival and was on Intravenous hydration and Intravenous antibiotics but he expired three days later during the course of treatment. As the case was of unnatural death, autopsy was performed.

External examination showed multiple ulcers with a leathery base and greenish mucoid deposit at the margins of ulcers, present over the chest, neck and near the right angle of mouth involving the both lips. Other areas of body showed whitish excoriated lesions. Yellowish discolouration of sclera and mucus membranes of oral cavity was seen. Internally both pleural cavities were filled with about one litre of straw coloured fluid. Both lungs were adherent to chest wall and congested. The heart was enlarged, liver was grossly congested, and both kidneys exhibited congestion with distorted cortico-medullary anatomy and stomach wall revealed congestion.

On scrutiny of these suspicious skin findings, the relatives were questioned again who stated that: the patient was a chronic alcoholic and one night attempted to drink paraquat that was used in their farm which accidently spilled on himself in a considerable amount. Following the incident, he stayed at home for four days lying on his bed wearing the same clothes, with mild complaints of chest pain and breathlessness and when symptoms worsened he was rushed to the hospital. The whitish skin lesions were explained to be present since a long time for which he was taking herbal medications. The police later acquired the bottle of Gramoxone from the site of alleged incidence.

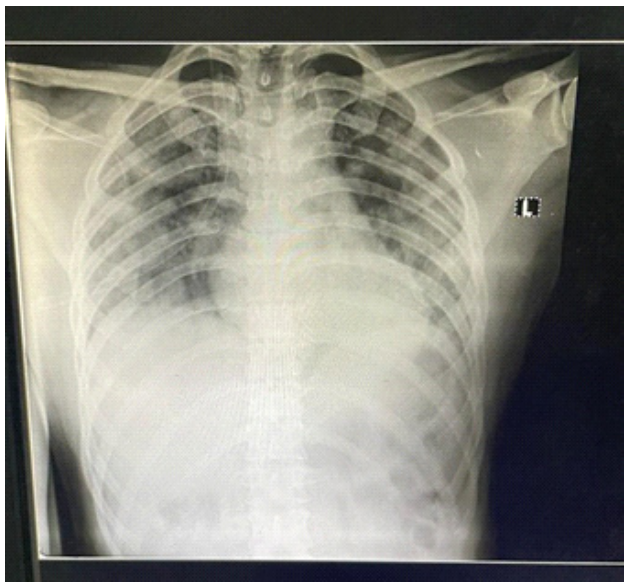

Figure 1: Chest X-ray on admission: Scattered non homogeneous

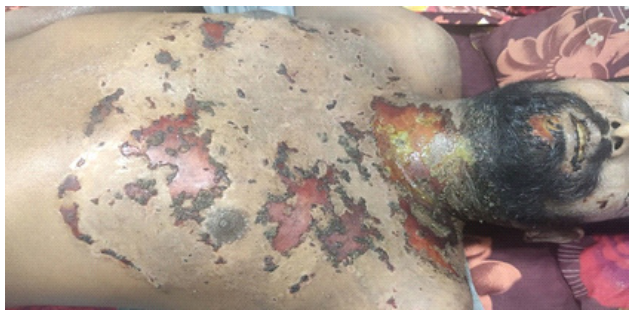

Figure 2: Reddish ulcerated lesions with leathery base

Figure 3: The t-shirt being worn since about seven days

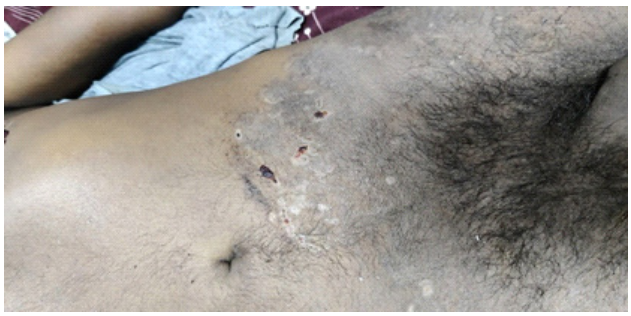

Figure 4: Whitish lesions over groin area

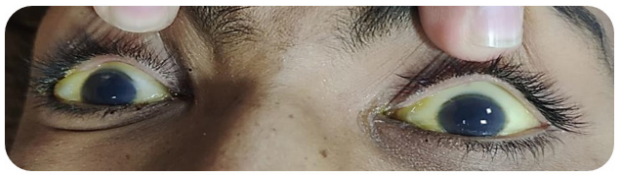

Figure 5: Icterus

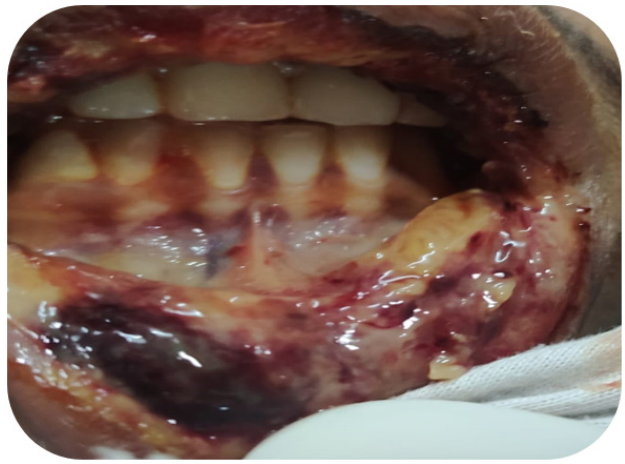

Figure 6: Closer image of the lip region Ulcers present in inner surface of lips

Discussion

Paraquat poisoning remains a significant worldwide cause of morbidity and mortality. Experiments have shown low percutaneous absorption of the paraquat through intact skin[6] this is the reason absorption of paraquat is scant with unblemished skin however instances of serious harmfulness have been seen in subjects with widespread skin damage.[7] Therefore, for significant absorption of the paraquat, damaged skin is essential. It can also aggravate itself any previous skin lesions present on the body. Even than few cases of death and extensive systematic involvement have been reported by authors and they have suggested low percutaneous permeability of the parquet through the intact skin and a damaged skin is essential for significant paraquat absorption.[4] Previous skin lesions can be aggravated by paraquat itself.[4-6] A case has been described in 1978 involving exposure of abraded skin to parquet that eventually results in respiratory failure.[8]

Although the exact cause of paraquat poisoning is unknown, it is usually assumed to be linked to oxidative stress and inflammation, When paraquat is absorbed via the skin, significant quantities of nicotinamide adenine dinucleotide phosphate (NADPH) are consumed, resulting in production of superoxide anions (O_2-), hydroxyl free radicals (HO–), and hydrogen peroxide (H_2O_2) and other

reactive oxygen species (ROS)inducing lipid peroxidation, this restricts breathing, and the normal electron transport chain, which results in mitochondrial damage and cell death. The effector cells produce a significant number of inflammatory factors as paraquat enters the lungs, amplifying the inflammatory response through the "cytokine cascade effect".[9] The histopathological findings vary from extensive pulmonary fibrosis in fatal cases to congestion, oedema, and haemorrhage in non-fatal cases. Diffuse consolidation of lungs is the initial pulmonary manifestation, and this progresses into cystic lesions after several days with very high mortality.[10,11]

In our case the patient, remained in contact to paraquat through the humid clothes with underlying broken skin. Oral ingestion was less as compared to the skin spillage which added to the delayed systemic toxicity. Major effects of poisoning are corrosion of the gastrointestinal tract, renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis and death is likely within several hours to a couple of days with features of vomiting, diarrhoea, fluid loss, shock, coma, convulsions, cardiac, hepatic and renal failure, and typically, pulmonary oedema. If hepato-renal failure does not cause quick death, gradual lung damage may cause mortality within the next two weeks. Subacute poisoning occurs when the pulmonary involvement occurs 24–48 hours after consumption.[12] In conclusion, it is clear that even minimal dermal exposure to paraquat can be fatal, especially when associated with high concentrations of the substance and/or significant delay in treatment initiation.[4]

Conclusion

Delayed contact with paraquat solutions even at low concentrations can cause systemic poisoning which may be fatal. The skin should be damaged in some way in order to allow adequate absorption of poison to be fatal. Although gastric lavage, haemodialysis and immunosuppressive agents are treatment choices but one has to note the importance of washing the contaminated skin, including hair and nails, vigorously, with soap following a dermal exposure. These deadly effects of the poison even on minimal exposure highlight the importance of use of protective clothing like PPE by the farmers. A thorough history taking is also essential along with examination by the caregivers to identify the poison and route of absorption to minimise the damage efficiently.

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Conflict of Interest: Nil.

Abbreviations:

ARDS- Acute Respiratory Distress syndrome.

PPE- Personal Protective Equipment.

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